LETTERS TO THE EDITOR

Carbamazepine in Reiter's syndrome

EDITOR,—A psoriatic spectrum with Reiter's syndrome as the most severe manifestation occurs with greater frequency in HIV infected individuals.[1] Immunosuppressive therapies for RS are associated with a poor response and increased morbidity.[2] We describe a case where carbamazepine showed an excellent response in an HIV infected patient with Reiter's syndrome.

A 30 year old married man presented with erythematous papules and plaques of 2 months' duration covered with hard limpet-like scales on face, body, and both extremities (fig 1). Palms and soles showed keratoderma blenorrhagicum and subungual hyperkeratosis with distal onycholysis. Both knees and wrists had painful swelling with restriction of movements. With this clinical presentation Reiter's syndrome was inferred. All routine investigations were normal except a raised erythrocyte sedimentation rate of 100 mm in the first hour. *x* Rays of the affected joints were normal. ELISA for HIV-1 and HIV-2 was positive with two kits (Immunocomb, Tri-dot) and confirmed with western blotting technique (Speciality Ranbaxy Limited). The absolute helper T lymphocyte count was 435 cells $\times 10^6$/l. Human leucocyte antigen B27 and rheumatoid factor were negative. The patient was commenced on prednisolone by mouth 60 mg daily and indomethacin by mouth 25 mg three times daily without any concomitant antiretroviral therapy. New erythematous papules and plaques appeared with no relief in joint pain and swelling.

In seeking an effective treatment, we serendipitously came across the efficacy of carbamazepine in an HIV infected patient with psoriatic erythroderma.[3] We started carbamazepine 200 mg daily in two divided doses in addition to above. The erythema cleared rapidly within 7 days. To confirm the effect of carbamazepine, it was stopped. New lesions similar to the old ones appeared within 3–4 days. Carbamazepine was then reintroduced in the same dose. Erythema cleared again within 7 days followed by scaling and joint swelling and pain. New lesions stopped appearing. Prednisolone was then tapered off rapidly and analgesics were stopped. Carbamazepine was continued in the same dose for 6 months. On follow up at 1 year, the patient showed no recurrence of skin lesions and synovitis, no change in liver and renal function tests, with no further deterioration in his overall health and no opportunistic infections.

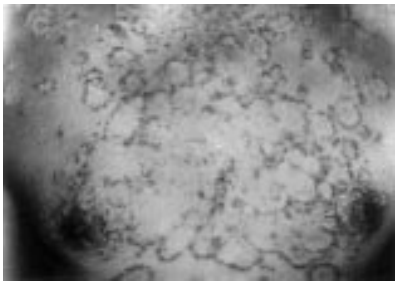

Figure 1 Close view of erythematous annular papules and plaques on chest before carbamazepine therapy.

It has been proposed that in genetically predisposed people, the release of neuropeptides like substance P, calcitonin gene related peptide, vasoactive intestinal peptide, and the inflammatory leucotriene B4 from cutaneous sensory nerves causes local inflammatory responses that trigger psoriasis.[4] Stimulated mast cells secrete a number of proinflammatory cytokines and proteases that act similarly.[4 5]

Carbamazepine significantly inhibits the uptake of noradrenaline (norepinephrine) and blocks a cyclic AMP mediated calcium influx that is associated with neuropeptide release and control of a slow potassium current.[6]

The rapid clearing of erythema, secondary to raised levels of neuropeptides, with carbamazepine may have been mediated through inhibition of these neuropeptides and by inhibition of uptake of noradrenaline. The exacerbation and subsequent resolution of lesions on withdrawal and reinstitution of carbamazepine respectively proves its efficacy in our patient. Also, the clinical remission maintained for 1 year after stopping carbamazepine confirms its therapeutic role in Reiter's syndrome. The therapeutic response seen in our patient conforms to that seen in the HIV-1 positive patient of Smith *et al*.[3]

This apparent success adds carbamazepine to the armamentarium against Reiter's syndrome in an HIV infected patient. This is the first reported case and an evaluation of long term carbamazepine therapy is warranted.

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Condoms and warts

EDITOR,—Wen *et al*[1] should be applauded for their attempt to address the key question of whether or not condoms protect people from genital warts. However, some of the major study variables need clarifying, as they did not match up with my knowledge of the Sydney Sexual Health Centre (SSHC) database.

The article discussed the issue of "acquisition of genital warts" and was presented as an incidence study. Cases were defined as: "All patients with a new diagnosis of macroscopic genital warts who attended SSHC [in] 1996." However, many of these patients had been previously diagnosed with genital warts elsewhere while others had recurrent lesions. In Australia, most genital warts are managed by general practitioners.[2] Consequently, the experience of specialist services is biased towards recurrent and difficult cases. "New diagnosis" in this situation means new to the clinic but not necessarily new to the patient. This means that the main outcome measure was a mixture of incident, prevalent, and recurrent cases, with the possibility that the warts may have affected the behaviour of many of the study subjects.

The SSHC database does document whether a person has previously been diagnosed with HPV infection. To me, the study would have had more validity if patients with a past history had been excluded.

The diagnostic grouping for warts at SSHC does not distinguish between genital and anal lesions. The readers of the journal need to know that many of these male "genital wart" cases would have been homosexually active men with anal warts. This is important as risk factors for penile and anal warts may differ, potentially confusing the results of the present study.

Originally developed as an HIV risk measure, the condom use variable at SSHC only refers to the previous 3 months or since the last registration/disease episode. Wen *et al*'s article[1] failed to mention that this variable was time limited. As 3 months is the median duration before the appearance of exophytic warts,[3] up to half of the relevant sexual behaviour may have been overlooked.

Finally, the referent group in the table describing condom use deemed as "Not applicable, no sex" should have been more accurately described as "No vaginal or anal sex in the previous 3 months." Many of these people would have practised oral sex or other sexual acts during those 3 months. Others may have ceased practising vaginal or anal intercourse up to 3 months earlier because of their persistent or recurrent warts.

Large relational quality assured clinical databases can be powerful tools for health service evaluation, surveillance, and the generation of research questions. It may be prudent for researchers to engage the people responsible for designing and maintaining those databases to minimise errors of interpretation.

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Reply

EDITOR,—We are grateful to Dr Dayan for her helpful and constructive comments. The major criticism of our paper relates to the selection of cases, and the possible inclusion